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Effect of Diabetes and Smoking on Salivary IgA levels in Stage III Grade A periodontitis patients - A Prospective Clinico-Biochemical Study

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Abstract

Background: Periodontitis is a chronic inflammatory disease affecting the supporting structures around teeth. Salivary immunoglobulin is thought to play an important role with non-inflammatory, pro-inflammatory and anti-inflammatory functions and is considered as the first line of defense.

Aim: Aim of the present study is to evaluate in stage III Grade A periodontitis subjects before and after SRP by estimating salivary IgA levels (by ELISA kit)and correlating them in diabetic non-smokers, smoker non-diabetic, smoker diabetic, and non-smoker non-diabetic individuals.

Materials and method: A total of 120 subjects with stage III Grade A periodontitis were divided into four groups (30 in each group) Diabetic Non-smokers (Group I), Smoker Non-Diabetic (Group II), Smoker Diabetic (Group III), Non-Smoker Non-Diabetic (Group IV).Clinical parameters like PI, GI, PPD and CAL and salivary IgA levels were recorded before at base line and after SRP at the end of 4 weeks.

Results: PI, GI scores were statistically significant in all the groups from baseline to 4 weeks (p=0.00001). Statistically significant correlation was observed for PPD in Group I vs Group III (p=0.0463*), Group II vs Group III (p=0.0039*), Group III Vs Group IV (p=0.0002). Clinical attachment level scores were

statistically significant for Group II vs Group III (p=0.0109*), Group III Vs Group IV (p=0.0007*) at 4 weeks. The increase in salivary IgA levels was statistically significant in all the groups from baseline to 4 weeks (p=0.00001).

Conclusion: The results showed statistically significant increase in salivary IgA levels in all the groups from baseline to four weeks with highest IgA levels in diabetic nonsmokers and lowest in smoker non-diabetics at baseline.

Keywords: Diabetes Mellitus, Smoking, Salivary IgA, Periodontal Disease

Introduction

Periodontitis is a chronic inflammatory disease affecting the supporting structures around teeth. The main immunoglobulin in secretions, including saliva is s-IgA. It is thought to play an important role with non-inflammatory, pro-inflammatory and anti-inflammatory functions and is considered as the first line of defense. The disproportion between pro-inflammatory and anti-inflammatory cytokines stimulates the progression of periodontal lesions and stops the resolution capacity of the immuno-inflammatory process.[1,2] Saliva, which is a complex body fluid that is increasingly used for early diagnosis and detection of potential vulnerability to several diseases. Analysis of whole saliva provides a simple and non-invasive method of evaluating the role of salivary IgA levels in periodontal disease.[3] Studies[4,5] have suggested a protective role for s-IgA in patients with PD and low concentrations of s-IgA have been associated with severe forms of thisdisease.[6]

Smoking is a risk factor for many systemic conditions and periodontal diseases. Furthermore, smoking, both active and passive, has been shown to be connected to significantly increased risks of type 2 diabetes mellitus (DM).[7]Systemic alterations of the cellular and humoral immune responses to periodontal pathogens among smokers have been evaluated, including immunosuppression, exaggerated inflammatory cell responses, impaired neutrophils and reduced antibody production. Smokers have decreased levels of salivary antibodies IgA and serum IgG antibodies.[8]Diabetes and periodontal disease are thought to be associated biologically and a number of studies have proposed mechanisms to explain the relationship, including microvascular disease, changes in components of gingival crevicular fluid, changes in collagen metabolism, an altered host response, altered subgingival flora, genetic predisposition and nonenzymatic glycation. The level of neutrophils and circulating immune complexes (IgG, IgA) in the sera of diabetic patients with periodontitis were found to be significantly higher compared to control subjects.[9]Hence the present study was undertaken to evaluate the host response in stage III Grade A periodontitis subjects before and after SRP by estimating salivary IgA levels and correlating them in diabetic non-smokers, smoker non diabetic, smoker diabetic, and non-smoker non diabetic individuals.

Materials and Methods

The Prospective biochemical study was carried out on total of 200 patients diagnosed with stage III Grade A periodontitis after obtaining informed consent and institutional ethical committee clearance. Patient above 18 years having minimum of 20 teeth were included in present study with

- At least three teeth each quadrant of the mouth having probing depth ≥5 mm

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- Attachment loss of at least ≥3mm in at least three teeth in each quadrant and radiographic evidence of horizontal and/or vertical bone loss.
- Individuals who have not undergone professional oral prophylaxis during the last six months.
- Subjects who have not received any antibiotic therapy 6 months prior to the commencement of the study.
- Subjects with a history of upper respiratory diseases of recent occurrence (within 4 weeks), allergic disorders or autoimmune disorders, and immune compromised states (e.g., HIV positive, primary and secondary immunodeficiency disorders, malnutrition etc.). and those Subjects on corticosteroid medication, sialagogues or antisioalogogues, Former smokers and non-smokers were excluded from the study.

A total of 120 individuals fulfilling the inclusion and exclusion criteria were selected from outpatient section. After baseline examination all the Subjects were assigned into 4 groups (thirty in each group):

Group I: Diabetic Non-Smokers

Group II: Smoker Non-Diabetic

Group III: Smoker Diabetic

Group IV: Non-Smoker Non-Diabetic

After screening, the diabetic status was evaluated using glycosylated hemoglobin (HbA_{1C}) levels (≥6.5% Diabetic and < 6.5% Non-diabetic for newly diagnosed cases as per ADA guidelines).Furthermore, depending on their smoking status obtained from history they were segregated into smokers and non-smokers. Subjects were classified as smokers based on criteria established by the centre for disease control and prevention (CDC), "current smokers" were defined as those who had smoked 100 or more cigarettes over their lifetime and smoked at the time of interview.

Study design:

After the selection of the subjects, Clinical parameters like Plaque Index,[10]Gingival Index,[11] Probing Pocket Depth and Clinical Attachment Levels were recorded at baseline and after 4 weeks from baseline. The probing pocket depth and CAL was measured using UNC-15 periodontal probe. Measurements were done for all the teeth at 4 sites per each tooth (mid buccal, mesiobuccally, distobuccal, lingual). Readings were recorded to the nearest millimeter.All measurements were performed by one experienced periodontal examiner, allowing an intra-experimental comparison of the values. Percentage agreement with another examiner within 1 mm was >96%.For all the enrolled subjects thorough scaling and root planning was performed.

Saliva sample collection and storage:

Saliva samples were collected from subjects the day after clinical examination. Subjects were refrained from smoking, eating or drinking for 1-2 hours prior to the test session. About 5 ml of unstimulated whole saliva samples were collected from each subject by the spitting method i.e., saliva is allowed to accumulate in the floor of the mouth and the subject spits it out into the test tube every 60 seconds.

Samples were then centrifuged (3000rpm per 15 min) and supernatants were collected and stored at -20 °C for analyses at a later date. Two samples of saliva were taken one at baseline and at the end of 4 weeks after scaling and root planning. Total s-IgA levels were then determined using an enzyme-linked immunosorbent assay (ELISA) using commercially available ELISA kit from DRG ELISAS, Manufactured by DRG instruments GmbH, GERMANY, REF IM-SLV- 4636.

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Sample was prepared by diluting supernatant salivary liquid 1:20 with diluted assay buffer followed by gentle mixing and leaving for at least 5 minutes on a rotating shaker. This was further diluted 1:50 with diluted assay buffer.

Statistical Analysis

The data were analyzed using the SPSS-software 26.00 program (SPSS Inc., Chicago, IL, USA). The intra group comparison of plaque index scores and gingival index scores between Group I, Group II, Group III &Group IV at various study intervals was done using Wilcoxon matched t test & intergroup comparisons were done by using Kruskal Wallis ANOVA. The intra group comparison of probing depth, clinical attachment level scores, salivary IgA levels between Group I, Group II, Group III &Group IV at various study intervals was done using paired t test & intergroup comparison was done by using one way ANOVA. Differences were considered as statistically significant at $p<0.05$*.

Results

A total of 120 subjects with stage III Grade A periodontitis were selected for the study comprising of 30 subjects per each group, Diabetic Non-smokers (Group I), Smoker Non-Diabetic (Group II), Smoker Diabetic (Group III), Non-Smoker Non-Diabetic (Group IV). The reduction of these plaque scores was statistically significant in all the groups from baseline to 4 weeks (p=0.00001) (Table 1). The reduction of gingival index scores was statistically significant in all the groups from baseline to 4 weeks (p=0.00001) (Table 2).

Probing pocket depth scores between the groups at baseline was positively correlated but statistical significance was observed only between Group II vs Group III (p=0.0003*), Group III vs Group IV (p=0.0019) and at 4 weeks. Statistically significant correlation was observed for Group I vs Group III (p=0.0463*), Group II vs Group III (p=0.0039*), Group III Vs Group IV (p=0.0002) (Table 3).

Clinical attachment level scores at baseline between the groups was positively correlated but statistical significance was observed only between Group II vs Group III (p=0.0079), Group II vs Group IV (p=0.9982) Group III vs Group IV (p=0.0159) and at 4 weeks statistically significant correlation was observed for Group II vs Group III (p=0.0109*), Group III Vs Group IV (p=0.0007*) (Table 4).

The increase in salivary IgA levels was statistically significant (Table 5) in all the groups from baseline to 4 weeks (p=0.00001).Salivary IgA levels at baseline between the groups was positively correlated but statistical significance was observed only between Group I vs Group II (p=0.0391), Group I vs Group III (p=0.045). At 4 weeks and the difference of salivary IgA levels at various study intervals showed no statistically significant correlation in all groups.

Discussion

Immunoglobulins contribute to the inhibition of bacterial adherence and colonization, enhance bacterial phagocytosis, and help detoxify bacterial toxins and thus play a major role in the primary defense against bacterial infections.[12] For the host to maintain homeostasis within the oral cavity, three distinct but interrelated immune responses contribute to control the microbial challenge. These are the salivary and gingival tissue (local) and the serum (systemic) immune systems.[13] These immunological responses can be mediated by three related fluid compartments: Saliva, crevicular fluid and blood. Hence immunoglobulins, if present, should be detected in these fluid compartments.[14,15]

s-IgA is considered to be a major factor contributing to mucosal health and microbial defense with wide range of biological activities against pathogens and is believed to act as an immune barrier to prevent adherence and absorption of microbes and various other antigens to the mucosa. It serves an important effector function at mucous membrane surfaces which are the main entry sites for most pathogenic organisms. Because it is polymeric, secretory IgA can crosslink large antigens with multiple epitopes. Furthermore, s-IgA can neutralize intracellular
microbial pathogens within the epithelial cells and facilitate their exclusion into the lumen.[16,17]

In the present study the intragroup comparisons of salivary IgA levels showed increase in salivary IgA levels from baseline to 4 weeks after SRP in all four groups. These findings are in correlation with studies by Basu et al.[18] Tynelius-Bratthall and Ellen[19], Ebersole et al.[20]

Basu et al.[18] observed lower salivary IgA levels in periodontitis patients before oral hygiene therapy and the concentrations of these immunoglobulins after periodontal therapy was increased. The observed difference between the pre- and post-surgery IgA levels were attributed to: 1) In active periodontal disease the salivary IgA were to form large complexes and aggregates with products of bacterial plaque and exudates. IgA is known to form such aggregates and to complex with other proteins.2) Before treatment excessive saliva production due to periodontal inflammation could have caused dilution of IgA.

Tynelius-Bratthall and Ellen[19] showed elevated salivary and crevicular antibodies to periodontal pathogens after conventional gingivitis treatment. The results were attributed to gingivitis which may be associated with a numerical predominance of A. viscosus relative to health specific salivary IgA antibodies that may have been "consumed" (absorbed to the increased total available A. viscosus antigens) during gingivitis. Ebersole et al.[20] examined the serum response in a longitudinal manner following subgingival scaling and the increase in antibody titers peak 100 to 200 times after scaling. The reason for this was related to inoculation of microorganisms into the host tissues resulting from scaling. Later the therapy that includes surgery and antibiotics lead to a gradual decline in antibody. This decline in antibody titers takes about 1 year.

Reiff[21] observed that levels of salivary and serum IgA declined after Phase I therapy which was attributed to fact that the initial therapy elicited decrease in IgA concentration when local antigenic stimuli could be reduced effectively by both dentist and patient. The less severe the periodontal involvement the more consistent is the reduction in IgA concentration following initial preparation therapy for unstimulated whole saliva.Srinivasan[22] observed a modest decline in the immunoglobulin levels after phase1 therapy in chronic periodontitis subjects and attributed it to be because of removal of antigenic stimuli.

Increase in IgA levels in the present study could be because of 1) Transient rise in the blastogenic response. 2) Inoculation of the microorganism into the host tissues resulting from scaling can lead to elevated titers. 3) Elimination of the immunosuppressive microorganism. 4) Single visit scaling and root planning. 5) The degree of severity of periodontal disease process which will affect the ability of the patient and the dentist to remove the antigenic challenge of the
Periodontium. 6) Effectiveness of removal of the antigenic stimulus on the part of the dentist. 7) Variation

with respect to oral microorganisms present at time of sampling, ability of patient to maintain oral health. 8) Before treatment excessive saliva production due to periodontal inflammation could have caused dilution of IgA. 9) More severely involved cases cannot have all local etiologic factors removed by initial preparation only 10)In the present study, clinical parameters plaque index, probing depth were reduced but not completely eliminated in all groups suggestive of persistence of antigenic stimulus which may also be responsible for increase in salivary IgA levels after 4 weeks compared to baseline.

In the present study, at baseline salivary IgA levels were elevated in group I when compared to group IV which is in accordance with studies by Anil[23], Navalker and Bhoweer[24] who also observed elevated IgA levels in diabetics compared to controls. This increased immunoglobulin concentration may be attributed to the stimulation of the immune system by specific bacterial infection in diabetics.

In contrast to the above findings de- Almeida et al.[25] observed lower s-IgA levels in diabetic individuals, more-frequent and more-severe periodontal disease and a greater need for periodontal treatment as compared with non-diabetic patients, suggesting a protective role of s-IgA, to be effective, require the cooperation of other host-protective mechanisms which are compromised in diabetics. Intergroup comparison of salivary IgA levels at baseline between group II and group IV showed lower IgA levels in group II compared to group IV but the difference was not statistically significant which is in accordance with the studies conducted by Olayanju et al.[26], Zuabi et al.[27]Zuabi et al.[27] evaluated the effect of smoking on periodontal status in subjects with established periodontitis, in contrast to the present study they observed that the difference in reduction in plaque index scores from first visit to second visit in smokers when compared to non-smokers was not statistically significant but the intergroup comparison of plaque scores at baseline observed was similar to that of the present study.

Reduction in gingival index scores from baseline to four weeks were not statistically significantly different between group I, IV which is in accordance with the studies by Galhardo Camargo et al.[28] and Mohan et al.[29].Reduction of gingival index scores from baseline to 4 weeks did not show a statistically significant difference between group II,IV which is in accordance with the study by Zuabi et al.[27] who observed no statistically significant difference in reductionof gingival index scores at various study intervals following non-surgical periodontaltherapy (SRP) in smokers when compared to non-smokers.

Gingival index scores at 4 weeks showed statistically significant difference between group II and IV which is in accordance with the study by Swathi et al.[30].Intergroup comparison of probing pocket depth between groups I and IV showed difference at baseline but was not statistically significant which is in accordance with the study by Christgau et al.[31] who observed no significant differences of the clinical parameters between diabetics and controls at baseline. In contrast Navarro-Sanchez[32] observed significantly greater pocket depth in diabetics whencompared to non-diabetics at baseline.

In the present study the CAL values in all the four groups exhibited gain in CAL from baseline to four weeks which was statistically significant within the groups which is in accordance with studies[31,32,33] in group I, IV. To the best of our knowledge, this is the

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first study to compare four groups that is diabetic nonsmoker, smoker non diabetic, smoker diabetic and non-smoker non diabetic with respect to both clinical parameters and IgA levels in saliva before and after SRP. Limitations of the study:1) Diabetic subjects were not categorized based on their metabolic control (controlled or uncontrolled). 2) Single visit scaling and root planning was performed. 3) Periodontitis subjects were not classified into mild, moderate, severe. 4) Long term follow up would have given a better idea of fluctuations in IgA levels following SRP.

Conclusion

The results showed statistically significant increase in salivary IgA levels in all the groups from baseline to four weeks with highest IgA levels in diabetic nonsmokers and lowest in smoker non diabetics at baseline. The initial phase of periodontal therapy, scaling and root planning though considered gold standard, the complete removal of the bacterial deposits and their toxins from the root surfaces within the periodontal pockets is not always achieved. So, increase in salivary IgA levels post therapy was thought to be due to the persistence of antigenic stimulus.

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Legend Tables

Groups	Baseline		4weeks		Difference		
	Mean	SD	Mean	SD	Mean	SD	P value
Group I	1.63	0.38	0.57	0.22	1.12	0.40	0.00001*
Group II	1.81	0.41	0.49	0.16	1.39	0.31	0.00001*
Group III	1.71	0.22	0.80	0.33	1.11	0.36	0.00001*
Group IV	1.88	0.12	0.59	0.21	1.21	0.13	0.00001*
F-value	2.2133		4.3421		7.1771		
p-value	0.5229		0.2290		0.0681		
Group I vs Group II	p=0.2191		p=0.9246		p=0.0620		
Group I vs Group III	p=0.7769		p=0.4188		p=0.8299		
Group I vs Group IV	p=0.3932		p=0.1239		p=0.4299		
Group II vs Group III	p=0.3299		p=0.5881		p=0.0214*		
Group II vs Group IV	p=0.5341		p=0.0192*		p=0.0488*		
Group III vs Group IV	p=0.3739		p=0.5981		p=0.2847		

*p<0.05

Table 1: Intra and Inter group comparison of four groups (I, II, III, and IV) with respect to plaque index scores at baseline and 4 weeks by Wilcoxon matched t test and Kruskal Wallis ANOVA respectively.

Groups	Baseline		4weeks		Difference		
	Mean	SD	Mean	SD	Mean	SD	P value
Group I	1.79	0.38	0.59	0.28	1.21	0.21	0.00001*
Group II	1.61	0.49	0.53	0.18	1.15	0.60	0.00001*
Group III	1.69	0.41	0.69	0.41	1.11	0.21	0.00001*
Group IV	1.89	0.26	0.79	0.21	1.19	0.23	0.00001*
F-value	11.6993		20.2890		1.9871		
p-value	0.0070*		0.00001*		0.5851		
Group I vs Group II	p=0.0686		p=0.0743		p=0.7355		
Group I vs Group III	p=0.1274		p=0.4920		p=0.2109		
Group I vs Group IV	p=0.4191		p=0.0399*		p=0.1712		
Group II vs Group III	p=0.4831		p=0.2233		p=0.6750		
Group II vs Group IV	p=0.0070*		p=0.00001*		p=0.6672		
Group III vs Group IV	p=0.0036*		p=0.0027*		p=0.8177		

*p<0.05

Table 2: Intra and Intergroup comparison of four groups (I, II, III, and IV) with respect to gingival index scores at baseline and 4 weeks by Wilcoxon matched t test and Kruskal Wallis ANOVA respectively.

Groups	Baseline		4weeks		Difference		
	Mean	SD	Mean	SD	Mean	SD	P value
Group I	3.79	0.63	3.12	0.79	0.82	0.41	0.00001*
Group II	3.49	0.81	2.93	0.62	0.59	0.60	0.00003*
Group III	4.50	0.84	3.76	0.82	0.85	0.38	0.00001*
Group IV	3.64	0.33	2.79	0.43	0.92	0.33	0.00001*
F-value	7.9391		7.3301		2.2791		
p-value	0.0001*		0.0002*		0.0872		
Group I vs Group II	p=0.2072		p=0.8249		p=0.3299		
Group I vs Group III	p=0.0586		p=0.0463*		p=0.9981		
Group I vs Group IV	p=0.5791		p=0.2970		p=0.8599		
Group II vs Group III	p=0.0003*		p=0.0039*		p=0.3971		
Group II vs Group IV	p=0.8982		p=0.8029		p=0.0611		
Group III vs Group IV	p=0.0019*		p=0.0002*		p=0.7732		

*p<0.05

Table 3: Intra and Intergroup comparison of four groups (I, II, III, IV) with respect to pocket depth scores at baseline and 4 weeks by paired t test and one way ANOVA respectively.

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Groups	Baseline		4weeks		Difference		
	Mean	SD	Mean	SD	Mean	SD	P value
Group I	4.22	0.62	3.49	0.63	0.59	0.32	0.00001*
Group II	3.65	0.59	3.29	0.59	0.59	0.27	0.00001*
Group III	4.53	1.12	4.18	0.91	0.69	0.39	0.00001*
Group IV	3.81	0.56	3.13	0.81	0.94	0.25	0.00001*
F-value	4.5301		6.1563		2.3581		
p-value	0.0051*		0.0007*		0.0789		
Group I vs Group II	p=0.4964		p=0.6629		p=0.9481		
Group I vs Group III	p=0.2519		p=0.1741		p=0.9573		
Group I vs Group IV	p=0.6243		p=0.2039		p=0.2978		
Group II vs Group III	p=0.0079*		p=0.0109*		P=0.9981		
Group II vs Group IV	p=0.9982*		p=0.8377		p=0.1049		
Group III vs Group IV	p=0.0159*		p=0.0007*		p=0.1331		

*p<0.05

Table 4: Intra and Intergroup comparison of four groups (I, II, III, IV) with respect to clinical attachment level scores at baseline and 4 weeks by paired t test and one way ANOVA respectively.

Groups	Baseline		4weeks		Difference		
	Mean	SD	Mean	SD	Mean	SD	P value
Group I	291.11	172.66	1297.31	706.79	989.17	673.69	0.00001*
Group II	178.69	119.59	1199.51	699.41	1019.91	679.11	0.00001*
Group III	199.11	129.69	1099.52	589.36	889.39	549.69	0.00001*
Group IV	209.71	119.21	1369.91	729.29	1171.19	731.60	0.00001*
F-value	2.8271		0.6389		0.5792		
p-value	0.0449		0.5981		0.6401		
Group I vs Group II	p=0.0391*		p=0.9691		p=0.9998		
Group I vs Group III	p=0.0456*		p=0.7883		p=0.9589		
Group Ivs Group IV	p=0.2039		p=0.9830		p=0.8529		
Group II vs Group III	p=0.9391		p=0.9649		p=0.9289		
Group II vs Group IV	p=0.8769		p=0.8391		p=0.8991		
Group III vs Group IV	p=0.9989		p=0.5618		p=0.5621		

*p<0.05

Table 5: Intra and intergroup comparison of four groups (I, II, III, and IV) with respect to IgA scores at baseline and 4 weeks by paired t test and one way ANOVA respectively.

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